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| APPLICATION NO.  | FILING DATE | FIRST NAMED INVENTOR | ATTORNEY DOCKET NO.            | CONFIRMATION NO. |
|--|-------------|----------------------|--------------------------------|------------------|
| 09/608,890   | 06/30/2000  | Gary L. Johnson      | CPI-004DVCP3CN                 | 1962             |
| 959  | 7590        | 11/01/2006           |                                |                  |
| LAHIVE & COCKFIELD, LLP<br>ONE POST OFFICE SQUARE<br>BOSTON, MA 02109-2127 |             |                      | EXAMINER<br>BASI, NIRMAL SINGH |                  |
|  |             |                      | ART UNIT                       | PAPER NUMBER     |
|  |             |                      | 1646                           |                  |

DATE MAILED: 11/01/2006

Please find below and/or attached an Office communication concerning this application or proceeding.

**Office Action Summary**

Application No.

09/608,890

Applicant(s)

JOHNSON, GARY L.

Examiner

Nirmal S. Basi

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-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

**Period for Reply**

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

**Status**

- 1) ☒ Responsive to communication(s) filed on 07 August 2006.
- 2a) ☐ This action is **FINAL**. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

**Disposition of Claims**

- 4) ☒ Claim(s) 53-58,66 and 67 is/are pending in the application.
- 4a) Of the above claim(s) \_\_\_\_\_ is/are withdrawn from consideration.
- 5) ☐ Claim(s) \_\_\_\_\_ is/are allowed.
- 6) ☒ Claim(s) 53-58,66 and 67 is/are rejected.
- 7) ☐ Claim(s) \_\_\_\_\_ is/are objected to.
- 8) ☐ Claim(s) \_\_\_\_\_ are subject to restriction and/or election requirement.

**Application Papers**

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on \_\_\_\_\_ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.  
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).  
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

**Priority under 35 U.S.C. § 119**

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some \* c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
  2. ☐ Certified copies of the priority documents have been received in Application No. \_\_\_\_\_.
  3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

\* See the attached detailed Office action for a list of the certified copies not received.

**Attachment(s)**

- |  |   |
|--|---|
| 1) <input checked="" type="checkbox"/> Notice of References Cited (PTO-892)  | 4) <input type="checkbox"/> Interview Summary (PTO-413)<br>Paper No(s)/Mail Date. _____ |
| 2) <input type="checkbox"/> Notice of Draftsperson's Patent Drawing Review (PTO-948)   | 5) <input type="checkbox"/> Notice of Informal Patent Application                       |
| 3) <input checked="" type="checkbox"/> Information Disclosure Statement(s) (PTO/SB/08)<br>Paper No(s)/Mail Date <u>4/15/03</u> . | 6) <input type="checkbox"/> Other: _____  |

### DETAILED ACTION

1. Amendment filed 8/7/06 has been entered.
2. It is noted due to a typographical error the cover sheet in the previous "Office Action Summary" indicated the Office Action as both "Final" and "Non-final". For clarification purposes the last Office Action was non-final.
3. The amended claims are newly rejected for the reasons given below, prior rejections are withdrawn.

### *Claim Rejections - 35 USC § 112*

The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

4. Claims 53-58 and 66-67 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method for regulating apoptosis of a cell comprising contacting the cell with an agent that directly interacts with the MEKK1 polypeptide set forth <sup>as</sup> a SEQ ID NO:2 or 4, such that apoptosis of the cell is regulated, wherein the agent that directly interacts with the MEKK1 polypeptide is one of the known ras oncogene proteins, <sup>regulating apoptosis</sup> does not reasonably provide enablement for ~~the use of~~ <sup>with</sup> ~~any cell~~ other agents. The, specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims.

The claims are drawn to a method for regulating apoptosis of a cell by contacting the cell with an agent that directly interacts with the MEKK1 polypeptide set forth a SEQ

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ID NO:2 or 4 such that apoptosis of the cell is regulated. The specification discloses one agent, ras oncogene protein (Rasv12), which directly interacts with the MEKK1 polypeptide and regulates apoptosis. The MEKK1 protein has been disclosed to have a regulatory domain (amino acid residues 1-408 of SEQ ID NO:2) and a kinase domain (about amino acid residues 409-672 of SEQ ID NO:2). The specification, page 110, discloses, "MEKK1 is a Ras effector and selectively binds to Ras in a GTP dependent manner. In addition, the binding of MEKK1 to Ras *in vitro* is direct and occurs via the COOH terminal region of MEKK1 that encodes the catalytic kinase domain. Page 107, first paragraph, discloses Ras can stimulate MEKK activity and that MEKK1 physically binds to Ras in a GTP-dependent manner. Page 109, discloses "GST-RasV12 binds to MEKK1 at a site located within the COOH- terminal catalytic domain of MEKK1". Page 110, first paragraph and Example 24 discloses purified recombinant MEKK1 protein bind directly to GST-Rasv12. There is no disclosure of any agents that interact with the regulatory domain of MEKK1 to regulate cell apoptosis. Although experiments are disclosed using Beauvericin to induce apoptosis there is no support in the specification or prior art that shows that it directly interacts with MEKK1 to exert its effect. Further, EGF, NGF and TPA stimulation of MEKK is shown (figs 5, 6 and 10), there is no support in the specification or prior art that indicates that EGF, NGF and TPA directly interact with MEKK1, their actions are by an indirect mechanism. The specification uses a number of established cell lines such as NIH 3T3 cells and PC12 cells to study MEKK1 interaction and apoptosis.

It must be noted that the specification does not show a single experiment where cell apoptosis is regulated by **contacting a cell** with an agent that **directly interacts** with the MEKK1 polypeptide. The ras oncogene proteins do not appear to traverse the cell membrane. Prior art discloses that microinjection of the ras oncogene protein into established cell lines *in vitro* regulates apoptosis (see Dafna Bar-Sagi, Ras Proteins: Biological Effects and Biochemical Targets, Anticancer Research Vol. 9, pages 1427-1438, 1998; Trahey et al, Biochemical and Biological Properties of Human N-ras p21 Protein, Molecular and Cellular Biology, Vol. 7, No. 1, pages 541-544, Jan 1987; Stacey et al, Transformation of NIH 3T3 Cells by Microinjection of Ha-ras p21 Protein, Nature, Vol. 310 pages 508-511, August 9, 1984; Lacal et al, Rapid Stimulation of Diacylglycerol Production in Xenopus Oocytes by Microinjection of H-ras p21, Science, new Series, Vol. 238, No. 7826, pages 533-536, October 1987). Cell lines used in the prior art that establish that ras oncogene proteins can regulate apoptosis include NIH 3T3 cells and PC12 cells (see references cited above).

The instant fact pattern for the claimed invention is similar to that in *In re Hyatt*, 708 F.2d 712, 218 USPQ 195 (Fed. Cir. 1983), wherein a single means claim which covered every conceivable means for achieving the stated purpose was held nonenabling for the scope of the claim because the specification at most disclosed only those means known to the inventors. When claims depend on a recited property, a fact situation comparable to *Hyatt* is possible, where the claim covers every conceivable structure or technique (means) for achieving the stated property (result) while the specification discloses at most only those known to the inventor. See also *Fiers v.*

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*Sugano*, 984 F.2d 164, 25 USPQ2d 1601 (Fed. Cir. 1993), and MPEP 2164.08(a). The specification discloses one agent that can interact in the manner required to regulate cell apoptosis. It is clear that highly specific agents and specific cells are required to practice the method as claimed. However, the claims fail to recite any specific agents or cell lines, and thus the skilled artisan would have to resort to trial and error experimentation to identify conditions meeting the functional limitations of the claims. Further the skilled artisan must discover by trial and error experimentation as to the signal transduction pathway of the agents as they relate to cell apoptosis. At the time of the invention, the state of the art established that known ras oncogene proteins could be microinjected into established cell lines (see references above, especially Trahey et al, column 1, first paragraph, page 541) to study their morphological differentiation. While many agents are known in the art that effect protein interactions it is not known which of these when contacted with a cell will traverse the cell membrane, directly interact with the kinase or regulatory domain of the MEKK1 protein and be capable of phosphorylating MEKK1 polypeptide, regulating the activity of a MEKK1 polypeptide substrate, controlling the phosphorylation of a signal transduction protein downstream of said MEKK1 polypeptide substrate, and regulating the activity of a signal transduction protein downstream of said MEKK1 polypeptide substrate, and predict which of the signal transduction proteins selected from the group consisting of MAPK, JNK and SAPK are directly involved in signal cascade initiated by agent binding to MEKK1.

Due to the large quantity of experimentation necessary to determine the specific agents that when contacted with a cell will traverse the cell membrane and directly

interact with the kinase or regulatory domain of the MEKK1 protein and have the signal transduction properties disclosed above, the lack of direction/guidance presented in the specification regarding the production of said agents, the absence of working examples directed to same, the complex nature of the invention, the state of the prior art which establishes the unpredictability and complex nature of the invention and the breadth of the claims which fail to recite any specific "agents", undue experimentation would be required of the skilled artisan to make and/or use the claimed invention in its full scope. Therefore, undue experimentation would be required of the skilled artisan to make or use the claimed invention in its full scope.

**Claim Rejection 35 USC 112, 1st paragraph (Written Description)**

5. Claims 53-58- and 66-68 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

The claims are drawn to use of a genus of undefined agents (no structure provided) which when contacted with a cell regulate apoptosis by directly interacting with the MEKK1 polypeptide set forth a SEQ ID NO:2 or 4 and is capable of phosphorylating MEKK1 polypeptide, regulating the activity of a MEKK1 polypeptide substrate, controlling the phosphorylation of a signal transduction protein downstream of said MEKK1 polypeptide substrate, and regulating the activity of a signal transduction protein downstream of said MEKK1 polypeptide substrate, and predict which of the

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signal transduction proteins selected from the group consisting of MAPK, JNK and SAPK are directly involved in signal cascade initiated by agent binding to MEKK1.

The specification discloses one agent, ras oncogene protein (Rasv12), which directly interacts with the MEKK1 polypeptide and regulates apoptosis. The MEKK1 protein has been disclosed to have a regulatory domain (amino acid residues 1-408 of SEQ ID NO:2) and a kinase domain (about amino acid residues 409-672 of SEQ ID NO:2). The specification, page 110, discloses, "MEKK1 is a Ras effector and selectively binds to Ras in a GTP dependent manner. In addition, the binding of MEKK1 to Ras *in vitro* is direct and occurs via the COOH terminal region of MEKK1 that encodes the catalytic kinase domain. Page 107, first paragraph, discloses Ras can stimulate MEKK activity and that MEKK1 physically binds to Ras in a GTP-dependent manner. Page 109, discloses "GST-RasV12 binds to MEKK1 at a site located within the COOH-terminal catalytic domain of MEKK1". Page 110, first paragraph and Example 24 discloses purified recombinant MEKK1 protein bind directly to GST-Rasv12. There is no disclosure of any agents that interact with the regulatory domain of MEKK1 to regulate cell apoptosis. Although experiments are disclosed using Beauvericin to induce apoptosis there is no support in the specification or prior art that shows that it directly interacts with MEKK1 to exert its effect. Further, EGF, NGF and TPA stimulate of MEEK is shown (figs 5, 6 and 10), there is no support in the specification or prior art that indicates that EGF, NGF and TPA directly interact with MEKK1, their actions are by an indirect mechanism. The specification uses a number



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of established cell lines such as NIH 3T3 cells and PC12 cells to study MEKK1 interaction and apoptosis.

It must be noted that the specification does not show a single experiment where cell apoptosis is regulated by **contacting a cell** with an agent that **directly interacts** with the MEKK1 polypeptide. The ras oncogene proteins do not traverse the cell membrane. Prior art discloses that microinjection of the ras oncogene protein into established cell lines *in vitro* regulates apoptosis (see Dafna Bar-Sagi, Ras Proteins: Biological Effects and Biochemical Targets, Anticancer Research Vol. 9, pages 1427-1438, 1998; Trahey et al, Biochemical and Biological Properties of Human N-ras p21 Protein, Molecular and Cellular Biology, Vol. 7, No. 1, pages 541-544, Jan 1987; Stacey et al, Transformation of NIH 3T3 Cells by Microinjection of Ha-ras p21 Protein, Nature, Vol. 310 pages 508-511, August 9, 1984; Lacal et al, Rapid Stimulation of Diacylglycerol Production in Xenopus Oocytes by Microinjection of H-ras p21, Science, new Series, Vol. 238, No. 7826, pages 533-536, October 1987). Cell lines used in the prior art that establish that ras oncogene proteins can regulate apoptosis include NIH 3T3 cells and PC12 cells (see references cited above).

The structure of the agents used in the claims has clearly not been established. The skilled artisan could not readily make the agents required to practice the claimed method or even predict their structure. The court and the Board have repeatedly held (*Amgen Inc. v. Chugai Pharmaceutical Co. Ltd.*, 18 USPQ2d 1016 (CA FC, 1991); *Fiers v. Revel*, 25 USPQ2d 1601 (CA FC 1993); *Fiddes v. Baird*, 30 USPQ2d 1481 (BPAI 1993) and *Regents of the Univ. Calif. v. Eli Lilly & Co.*, 43 USPQ2d 1398 (CA FC,

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1997)) that an adequate written description of an agent requires more than a mere statement that it is part of the invention and reference to a potential method for isolating it, irrespective of the complexity or simplicity of the method; what is required is a description of the agent itself. It is not sufficient to define compound solely by its principal biological property, because disclosure of no more than that, as in the instant case, is simply a wish to know the identity of any agent with that biological property. Naming a type of material generically known to exist, in the absence of knowledge as to what that material consists of, is not a description of that material. When one is unable to envision the detailed constitution of a complex chemical compound having a particular function so as to distinguish it from other materials, as well as a method for obtaining it, conception has not been achieved until reduction to practice has occurred, i.e., until after the agent has been isolated. Thus, claiming all agents that achieve a result without defining what means will do so is not in compliance with the description requirement. Rather, it is an attempt to preempt the future before it has arrived. Also, where a claim purports to cover all agents that may have a particular function the specification discloses but a single agent, Ras, known to do so. The situation is analogous to a single means claim and does not meet the enablement requirement under paragraph 1 of 112. The court has also held that a claimed agent could meet the written description and enablement requirements if the agent were defined by a disclosed process found, after-the-fact, to produce the agent, and claimed as a product-by-process. However, in the instant case, the agent is not claimed as a product-by-

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process, nor does the specification disclose any process known to yield a claimed agents.

The only difference between the cases reviewed by the court and Board, and the instant case, is that in addition to recitation of the desired agent activity. The claims recite no structural relationships of the agent as it relates to function. The critical feature of the agent as it relates structure to function is not disclosed.

For example, if one skilled in the art were to make a synthetic agent he would not be able to predict if it would interact directly with MEKK1 to regulate apoptosis and effect any of the other proteins in the signal pathway.

The specification does not provide any information on the structural relationship of the agents as it relates to function. In *Amgen Inc. v. Chugai Pharmaceutical Co. Ltd.*, 18 USPQ2d 1016 (Fed. Cir. 1991), the court ruled that a claim to a large genus of possible genetic sequences encoding a protein with a particular function that needs to be determined subsequent to the construction of the genetic sequences may not find sufficient support under 35 USC 112, 1st paragraph if only a few of the sequences that meet the functional limitations of the claim are disclosed and if undue experimentation would be required of one skilled in the art for determining other genetic sequences embraced by the claim. This is the case here, where specification discloses only one putative functional agent, Ras oncogene protein, having the necessary properties for the disclosed uses, and provides no guidance on obtaining agents, which encompass agents with no defined structure.

The common function of the agents, which is based upon a common property or critical technical feature of the genus claimed is not disclosed. The claims, as written, encompass agents, which vary substantially in composition. The instant disclosure of ras oncogene product does not adequately describe the scope of the use of the claimed genus, which encompasses a substantial variety of subgenera. A description of a genus of agents may be achieved by means of a recitation of a representative number of agents, defined by structure, falling within the scope of the genus or of a recitation of the structural features common to members of the genus, which features constitute a substantial portion of the genus. See *Regents of the University of California v. Eli Lilly & Co.*, 119 F3d 1559, 1569, 43 USPQ2d 1398, 1406 (Fed. Cir. 1997).

Since the disclosure fails to describe the common attributes or characteristics that identify members of the genus, and because the genus may be highly variant, the disclosure of one agent is insufficient to describe the genus. One of skill in the art would reasonably conclude that the disclosure fails to provide a representative number of species to describe, enable and use the genus as broadly claimed. The skilled artisan cannot envision the detailed chemical structure of the encompassed proteins and, therefore, conception is not achieved until reduction to practice has occurred, regardless of the complexity or simplicity of the method of isolation. Adequate written description requires more than a mere statement that it is part of the invention and reference to a potential method for isolating it. It is acknowledged that the skill of the artisan in the biochemistry art is high. However, in the current instance, because of the lack of guidance in the prior art and current application, one skilled in the art could not

predict which agents have the same activity as claimed since the critical special technical feature of the claimed agent is disclosed.

The skilled artisan cannot envision the detailed chemical structure of the encompassed compounds and, therefore, conception is not achieved until reduction to practice has occurred, regardless of the complexity or simplicity of the method of isolation. Adequate written description requires more than a mere statement that it is part of the invention and reference to a potential method for isolating it. *Vas-Cath Inc. V. Mahurkar*, 19 USPQ2d 1111, clearly states that "applicant must convey with reasonable clarity to those skilled in the art that, as of the filing date sought, he or she was in possession of the invention. The invention is, for purposes of the 'written description' inquiry, *whatever is now claimed*." (See page 1117). The specification does not "clearly allow persons of ordinary skill in the art to recognize that [he or she] invented what is claimed." (See *Vas-Cath* at page 1116).

Applicant is reminded that *Vas-Cath* makes clear that the written description provision of 35 USC 112 is severable from its enablement provision (see page 115).

Adequate written description requires more than a mere statement that it is part of the invention and a reference to a potential method of isolating it. The agent itself is required. See *Fibers v. Revel*, 25 USPQ d. 1601 at 1606 (CAFC 1993) and *Amen Inc. V. Chugai Pharmaceutical Co. Lts.*, 18 USPQ2d 1016.

Furthermore, In *The Regents of the University of California v. Eli Lilly* (43 USPQ2d 1398-1412), the court held that a generic statement, which defines a genus of compounds by only their functional activity, does not provide an adequate written

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description of the genus. The court indicated that while Applicants are not required to disclose every species encompassed by a genus, the description of a genus is achieved by the recitation of a representative number of compounds falling within the scope of the claimed genus. At section B(1), the court states that "An adequate written description of a DNA...requires a precise definition, such as by structure, formula, chemical name, or physical properties', not a mere wish or plan for obtaining the claimed chemical invention". The ras oncogene product is enabled for use in claimed method the use of other agents rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

### **Claim Rejections, 35 U.S.C. 102**

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless --

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

6. Claims 53-58 and 66-67 are rejected under 35 U.S.C. 102(e) as being anticipated by Dafna Bar-Sagi (Ras Proteins: Biological Effects and Biochemical Targets, Anticancer Research Vol. 9, pages 1427-1438, 1989).

Dafna Bar-Sagi discloses that microinjection of the ras oncogene protein into established cell lines such as NIH 3T3 cells, PC12 (neuronal cancer cells), embryo fibroblasts regulates apoptosis. The PC12 cells were used in instant application to show MEKK1 and ras interaction. The PC12 cells inherently contain the MEKK1 because an endogenous MEKK1 activity immunoprecipitated from PC12 cells was recently found to be growth factor-regulated in a Ras dependent manner (Russell et al, Direct Interaction between Ras and Kinase Domain of Mitogen-activated Protein Kinase Kinase Kinase (MEKK1) JBC, Vol. 270, No. 20, pages 11757-11760, May 1995, see introduction). Russell et al further show, a) Mitogen-activated protein kinase kinase kinase (MEKK1) is a serine-threonine kinase that regulates sequential protein kinase pathways involving stress-activated protein kinases and mitogen-activated protein kinases, b) MEKK1 is activated in response to growth factor stimulation of cells and by expression of activated Ras, c) The kinase domain of MEKK1 (MEKK1) binds to GST-Ras in a GTP dependent manner, d) Purified bacterially expressed MEKK1 binds to GST-RasV12 GTP $\gamma$ S demonstrating a direct interaction of the two proteins, e) A Ras effector domain peptide blocks the binding of MEKK1 to GST-RasV12 (GTP $\gamma$ S). MEKK1 complexed with GST-RasV12 (GTP $\gamma$ S) is capable of phosphorylating MEK1. The teachings of Russell show that MEKK1 directly binds Ras GTP. Thus, Ras interacts with protein kinases of both the Raf and MEKK families. The findings of Russell et al are inherent to the method disclosed by Bar-Sagi.

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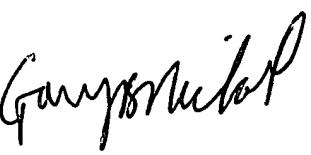
Therefore, due to the inherency of ras and MEKK1 interaction signal transduction pathway the method of Dafna Bar-Sagi anticipates claim 53-58 and 66-67, absent evidence to the contrary.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Nirmal S. Basi whose telephone number is 571-272-0868. The examiner can normally be reached on 9:00 AM-5:30 PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Gary Nickol can be reached on 571-272-0835. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

Nirmal S. Basi  
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10/26/06

*Niv*  
  
GARY B. NICKOL, PH.D.  
SUPERVISORY PATENT EXAMINER  
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